COMPARISON OF BLOOD LOSS IN ORTHOGNATHIC SURGERY WITH NORMAL AND HYPOTENSIVE ANAESTHESIA

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In partial fulfillment of the requirement for the degree of

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Certificate

This is to certify that **Dr. C. Dhandapani,** Post Graduates student (2007 – 2010) in the Department of Oral and Maxillofacial Surgery, Tamil Nadu Government Dental College and Hospital, Chennai has done this dissertation titled ***"Comparison of Blood loss in Orthognathic Surgery with normal and Hypotansive Anaesthesia"*** under my direct guidance and supervision in partial fulfillment of the regulations laid down by the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D.S., Branch - III Oral and Maxillofacial Surgery Degree Examination.

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DECLARATION

I, **Dr. C. DHANDAPANI,** do hereby declare that the dissertation titled **"COMPARISON OF BLOOD LOSS IN ORTHOGNATHIC SURGERY WITH NORMAL AND HYPOTENSIVE ANAESTHESIA"** was done in the Department of Oral and Maxillofacial Surgery. Tamil Nadu Government Dental College & Hospital, Chennai 600 003. I have utilized the facilities provided in the Government dental college for the study in partial fulfillment of the requirements for the degree of **Master of Dental Surgery** in the specialty of Oral and Maxillofacial Surgery (**Branch III**) during the course period **2007 – 2010** under the conceptualization and guidance of my dissertation guide, **Prof. Dr. D. Durairaj, M.D.S.**

I declare that no part of the dissertation will be utilized for gaining financial assistance for research or other promotions without obtaining prior permission from the Tamil Nadu Government Dental College & Hospital.

I also declare that no part of this work will be published either in the print or electronic media except with those who have been actively involved in this dissertation work and I firmly affirm that the right to preserve or publish this work rests solely with the prior permission of the Principal, Tamil Nadu Government Dental College & Hospital, Chennai 600 003, but with the vested right that I shall be cited as the author(s).

Signature of the PG student *Signature of the Guide*

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Introduction

Introduction

Various dentofacial deformities are corrected by orthognathic surgical procedures, which lead to significant blood loss due to soft tissue and intrabony bleeding from medium sized vessels to capillaries. Most of the bleeding cannot be controlled by usual surgical techniques employed in soft tissue surgery like identification and ligation of vessels. In this situation induced hypotensive anesthesia appears as an ideal method to decrease the blood loss and amount of transfusions following orthognathic surgery. The whole blood loss is decreased by more than 40% during hypotensive anesthesia as compared with normotensive anesthesia for similar orthognathic surgical procedures. Dropping the mean arterial pressure from preoperative level of 90mm of Hg to intraoperative level of 60mm of Hg effects this reduction in blood loss. It was decided to conduct a randomized, prospective clinical study to determine the efficacy of hypotensive anesthesia in minimizing blood loss during orthognathic surgery.

Deliberate Hypotension:

Deliberate or controlled hypotension is an anesthetic technique that permits the clinician to lower arterial pressure effectively in order to decrease blood loss during surgery and to provide a blood less field for the surgeon.

The main purpose of deliberate hypotension is to decrease blood loss, thereby decreasing the need for blood transfusion and, or improving operating conditions at the surgical site. The latter, namely improved visibility for the surgeon to perform more delicate surgical procedures. The bleeding could be controlled not only by a reduction in blood pressure, but by proper positioning of the patient as well. Since that time, deliberate hypotension has often been controversial, primarily because of inability to define the lowest safe arterial pressure with confidence. Such terms as "Controlled hypotension", "induced hypotension", "deliberate hypotension" and "hypotensive anesthesia" have all been used.

Review of Literature

Review of Literature

Controlled hypotension has been changing continuously and evolving not only as the result of the discovery of new agents and techniques, but also from an improved understanding of the underlying physiological changes which accompany its practice. Almost without exception, the techniques and agents used for controlled hypotension have arisen from an "accidental" finding.

DAVY (1800) [10] gave an enigmatic statement in the history of anesthesia stating if nitrous oxide is used with advantage during surgical operations there was no great effusion of blood.

In a few short years following a Second World War anesthesia took a second leap forward after a century of relative quiescence

KING (1934) [39] studied the activity of certain compounds which arose from the discovery of the quaternary nitrogen atoms in the structure of tubocurarine.

MINITT and GILLIES (1944) [50] showed that anesthesia was maintained with chloroform, cyclopropane or sometimes trichloroethelene together with nitrous oxide and oxygen.

GARDNER (1946) [22] described about hemorrhage and hypotension as a technique to control it.

GRIFFITHS and GILLIES (1948) [26] described hypotension by giving a high spinal block and showed a decrease in blood loss.

PATON and ZAIMIS (1948a) [56] set out to explore the tubocurrarine like action of polymethelene, bis-quaternary ammonium salts.

PATON and ZAIMIS (1948a) [56] wrote on the clinical potentialities of two members of the series of compounds namely hexamethonium and decamethonium.

HUNTER (1950) [34] considered hexamethonium as a more effective antidote than pentamenthonium.

DAVISON (1950) [9] and ORGANE (1950) [54] were deliberately lowering arterial pressure during anesthesia at New Castle and West Minister hospital, to reduce blood loss intraoperatively.

SCURR (1951) [66] introduced suxamethonium into clinical practice

GRAY and REES (1952) [25] used muscle relaxants and caused deliberate paralysis with artificial ventilation via an endotracheal tube. These caused narcosis, relaxation and reflex suppression.

WYMAN (1953) [81] gave details, based on a series of 1000 major surgical cases in which induced hypotension has been used.

ENDERBY (1953) [18] introduced pentolinium, a ganglionic blocking agent, which is short acting and to be given by infusion and thus permitting a new dimension in controllability which has been surpassed only by sodium nitroprusside.

MORACA et al (1962) [51] quoted that neurosurgical practice has an obvious requirement for a technique of extreme evanescence and for this reason introduced sodium nitroprusside into clinical practice, which caused hypotension intraoperatively.

ECKENHOFF et al (1963)[13] demonstrated enormous dead space during induced hypotension and most authorities advocate controlled ventilation to ensure adequate alveolar ventilation.

ECKENHOFF et al (1964) [14] did a comparative study on two groups one with hypotension and the other with normotension and found that deliberate hypotension did not lead to changes in mental function as measured by psychological tests.

GILLIES and HOLMES (1965) [23] with reference to the triad's approach explained that venous congestion associated with respiratory obstruction and the cardiovascular effects of carbon dioxide retention are the commonest causes of abnormal ozzing and hypotension is necessary to prevent these effects.

BROWN and HORTON (1966) [6] described a technique in which a cardiac pacemaker electrode was used to pace the heart so rapidly that stroke volume was extremely curtailed however, this had limited application.

SCURR (1971) [67] quoted that death from anesthesia passed its zenith in spite of an increase in the number of administrations.

ROLLASON, W.N. et al (1971) [64] did a comparative study of mental function in relation to hypotensive and normotensive anesthesia in the elderly and concluded that both groups of patients were equally at risk.

PRYS – ROBERTS et al (1972) [60] predicted the decrease in cardiac output associated with most techniques expect nitroprusside administration and the severe depressant effect of high doses of halothane on the myocardium was appreciated.

BLACKBURN et al (1973) [4] stressed the role of posture and an increased airway pressure in relation to pharmacological blockade during anesthesia.

HARP J. R and WOLLAMN H (1973) [30] explained the compensatory mechanism that tends to prevent cerebral hypoxia during controlled hypotension.

ECKENHOFF and LEIGH (1974) [15] demonstrated that adequate alveolar ventilation in induced hypotension is a must but without hyperventilation.

GRIFFITHS D.P.G et al (1974) [27] did a study of cerebral blood flow and metabolism during hypotension induced with sodium nitroprusside and found out that there were no complications that be attributed to sodium nitroprusside.

LINDOP (1975) [44] stated that controlled hypotension and shock have in common a low arterial pressure, and since the latter may be fatal and instinctive reaction, has always been that deliberate hypotension must of it self increase morbidity and mortaliy over non hypotensive techniques for the same surgical procedure.

* THOMPSON G. et al (1978) [75] previous study of blood loss and organ functions to determine the effect of induced hypotension postoperatively on brain, liver and kidney function and found that there was no morbidity and this procedure shortened the operating time, decreased blood loss and decreased the number of blood transfusions.

BEHNIA R. et al M.D (1978) [1] did a study on sodium nitroprusside induced hypotension and its effect on renal function and found out that medullary renal tissue oxygenation, and index of tissue viability, may remain adequate, despite a significant decrease in endogenous creatinine clearance during hypotension procedure.

* FAHMY N.R M.D (1978) [21] did a comparative study on nitroglycerine and nitroprusside and found out that nitroglycerine is an effective hypotensive drug that is proved superior as it is safe and does not

cause any myocardial infarction, renal damage, or cerebral vascular complications.

MURALI SIVARAJAN. M.D et al (1980) [52] did a study on what determines the blood loss during induced hypotension and found out that operative blood loss during induced hypotension is determined by mean arterial pressure and not cardiac output.

Chamberlain J.H et al (1980) [7] compared drug induced hypotension between halothane and nitroprusside and hypotension on myocardial metabolism and concluded that reduction in mean arterial pressure to those levels by either method is not likely to impair myocardial energetics in healthy hearts with normal coronary anatomy.

NORLEN K (1988) [53] did a study on central and regional hemodynamics during controlled hypotension produced by adenosine, nitroprusside, nitroglycerine and found out that urine flow was greatly impaired during the infusion of adenosine.

LESSARD M.R. et al (1989) [43] did a study on isoflurane-induced hypotension in orthonathic surgery and found out that isoflurane effectively reduced blood loss and the number of transfusions in orthognathic surgery.

YOSHITO SHIRAIHI et al (1994) [82] did a study on controlled hypotension and oxygen uptake and carbon dioxide elimination and found out that the balance between them and demand of oxygen supply was maintained during induced hypotension with PGE_1 or nitroglycerine.

PRECIOUS D. et al (1996) [58] compared blood loss, quality of surgical Field, and duration of procedure with and without induced hypotension anesthesia in adolescent orthognathic surgery patients and concluded that induced hypotensive anesthesia results in both reduced blood loss and improvement in surgical field.

Materials and Methods

Materials and methods

After obtaining approval from the patient and the parents, 6 patients within the age group of years 21-34 were studied in a prospective, randomized, stratified and single blind fashion.

The patients were randomly allotted to **A** or **B** group**.** **"A"** group is a normotensive group (normal) on whom a routine anesthetic procedure, is to be adopted**.** **"B"** group is a hypotension group on whom hypotensive anesthesia is to be adopted.

The patients selected for study were those requiring orthognathic procedures, for either single or both the jaws. All patients were healthy ASA class I subjects. The ASA class includes:

Class I: Healthy patient

Class II: Mild systemic disease – no functional limitation.

Class III: Severe systemic disease [a] – definite functional limitation.

Class IV: Severe systemic disease [a] that is constant threat of life.

Class V: Moribund patient not expected to survive 24 hrs with or

without operation.

(a – Whether or not the systemic disease is the disease for which a patient is undergoing surgery.)

Patients belonging to ASA II-IV were not included in the study. In particular asthmatic patients were strictly excluded. Preoperative investigations included hemoglobin percentage, packed cell volume, urinenalysis, urea and creatinine. The patient's baseline blood pressure was recorded.

Method of general anesthesia

Premedication: All the patients included in the study were given the following pre-medications:

8 Hours before surgery ---- Tab-Diazepam --------- 10mg

Tab-Ranitidine --------- 150mg

Tab-Metaclopromide --- 10mg

1 hour before surgery ------ Inj – Glycopyrrolate ----- 0.2mg I.M.

Preparation:

Two good venous accesses one of the infusion of hypotensive agents and the other for the fluid administration were gained. Inj – Cephatxim 1gm and Metronidazole 1gm were given as a routine prophylactic antibiotic. Inj – Dexamethasone 8mg was given as a routine. Bladder was catheterized with Folley's catheter to monitor hourly urine output.

The patients were connected to the monitors like continuous ECG> $SpO_{2,}$ Pulse rate. Respiratory rate and non-invasive blood pressure. The alarms were selected and entered into the monitor.

250ml of 5% dextrose was infused before induction.

Procedure

Procedure

The patient's surgical plan was determined, and a general pre – operative assessment was carried out before the surgical procedure. The patients included in the study were pre-medicated. The pre-operative blood pressure was recorded before the patient was shifted to the theatre.

Induction:

Anesthesia was induced with 5mg/kg of Thiopentone Sodium and Inj-Tramadol 2mg/kg was used as an analgesic agent for both the groups.

Intubation:

In patients with a normal predicted airway Vercuronium Bromide 0.05mg/kg were used as a muscle relaxant to facilitate the naso-endotracheal intubation. In patients with a predicted abnormal airway Inj. Succinylcholine was used as a muscle relaxant at 2mg/kg dose. The same method was adopted in both the groups.

Position:

Both the group patients after intubation were positioned in (10^0 Fowler position with a sandbag in the inter-scapular region.

Maintenance of Anesthesia:

In B group patients anesthesia was maintained with 70% of nitrous oxide, 30% of oxygen and 1% of halothane. Holothane was slowly weaned off and stopped at the time of would closure, In **'A'** – normotensive group of patients halothane of 0.5% was used for the initial hour and stopped.

A) Hypotensive agents: Used on B group – hypotensive patients.

1) Nitroglycerine - 50mg in 500ml normal saline was infused Through a volumetric infusion pump (Figure-1) just before the Induction and this NTG was titrated to keep the mean arterial pressures in between 60 -65mm of

Figure - I

2) Esmolol was used to prevent the tachycardia response by the nitroglycerine. Esmolol was started at 300 micrograms/kg as a bolus followed by an infusion rate to keep the heart rate less than 100 / min. This esmolol was infused in a syringe infusion pump (Figure-II)

Figure - II

B) Anesthetic agents:

In patients of both the groups

1) Vercuronium Bromide. a non-depolarizing muscle relaxant was used to maintain the muscle relaxation.

2) Tramadol was toped up at 1mg/kg dose if the surgery was prolonged For more than three hours.

C) Hypotensive level

The mean arterial pressure was maintained in between 60-65mm of Hg throughout the procedure until the surgeon fixed up the osteotomy segments. In case of reduced urine output of less than 0.5ml/kg/hr in spite of adequate fluid replacement, the mean arterial pressure was slowly raised roughly by around 10mm of Hg to retain the urine output. At the end of the segmental fixation, by reducing the NTG infusion rate to the pre operative value, the mean arterial pressure was slowly raised and the hemostasis checked.

D) Fluid management:

A strict fluid replacement protocol was followed. Ringer's lactate was infused intra-operatively at the rate of 6ml/kg/hr, with a volumertric pump to maintain routine intra-operative fluid requirements. Allowable blood loss (ABL) was defined as 20% of estimated blood volume (male 80ml/Kg, female 70ml/Kg). Blood loss upto the ABL replaced with 3ml of Ringer's lactate for each milliliter of blood loss. When blood losses reached the ABL, one unit of blood was transfused and then additional unit for every 400 ml of blood loss.

Monitoring:

It includes a non-invasive blood pressure monitor (Figure-III), E.C.G., Pulse oximeter and urinary output. Blood pressure both systolic, diastolic and mean arterial pressure, heart rate, SpO_2 were checked and recorded every fifteen minutes. Urine output was measured every hour.

Figure - III

Before the incision, the surgeon infiltrated the oral mucosa with 2% lignocaine with adrenaline 1:80000.

The duration of the anesthesia, from anesthetic induction to the reversal of muscle relaxant's effect was recorded. The time from first incision to the completion of the last suture placement was recorded as the surgical time. During the procedure, the amount of irrigation fluid was precisely measured.

Pharmacokinetics & Pharmacodynamics

PHARMACOKINETICS AND PHARMACODYNAMICS

Pharmacology of the hypotensive anesthetic drugs:

The drugs, used in deliberate hypotension, are:

1. Inhaled anesthetics - Halothane
2. Intravenous anesthetics - Nitroglycerinee
   - Esmolol

Halothane:

Chemical and Physical properties:

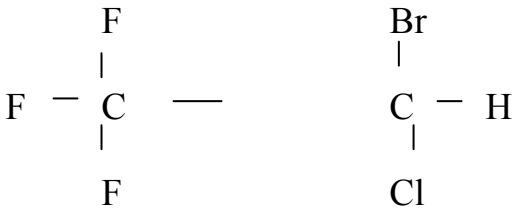
Halothane (fluothane) is 2-bromo-2chloro-1, 1-trifluoroethane. Mixtures of halothane with air or oxygen are not flammable or explosive.

With the exception of chromium, nickel, and titanium, most metals are tarnished or corroded by halothane. The compound interacts with rubber and some plastics, but not with polyethylene.

The solubility of halothane in rubber can theoretically slow the induction and the emergence from anesthesia as a consequence of the uptake

or release of the anesthetic from the rubber elements in the anesthesia circuit when low flow techniques are used.

The structure of halothane is :



Pharmacological properties:

It is a potent anesthetic agent with properties that allow a smooth and rather rapid loss of consciousness that progresses to anesthesia. However, the rapidity, convenience, and pleasantness associated with intravenous administration of thiopental are usually preferred for induction of anesthesia; halothane is then introduced for maintenance of anesthesia during the surgical procedure. The circumstances and requirements of the surgical procedure determine whether the trachea is intubated; whether the patient is allowed to breathe spontaneously or is ventilated manually or mechanically; and whether additional drugs, such as muscle relaxants or analgesics, are administered.

The signs of depth of anesthesia with halothane that are most practical value are the blood pressure, which is progressively depressed, and the response to surgical stimulation (e.g: pulse rate, blood pressure, movement or

even awakening). The concentration of anesthetic agent that is necessary in the inspired gas mixture for induction of anesthesia must be appropriately reduced as an alveolar concentration increases during maintenance if progressive increase in depth of anesthesia and decrease in blood pressure are to be avoided.

Circulation:

Administration of halothane is characterized by a dose dependent reduction of arterial blood pressure. Hypotension results from two main effects. First, the myocardium is depressed directly and cardiac output is decreased; second, the normal baroreceptor-mediated tachycardia in response to hypotension is obtunded.

Heart:

Then anesthesia is induced by inspiration of halothane at concentrations commonly necessary for surgical anesthesia (0.8 to 1.2%), cardiac output is reduced by 20 to 50% from the level characteristic of the awake state (Marshall et al 1969) [46]. Both increased concentrations of halothane and reduced arterial carbon dioxide tension (hyperventilation) accentuate the reduction in cardiac output.

The contractility of preparations of heart muscles in vitro is depressed by halothane in a dose dependent fashion. It is also generally agreed that myocardial contractility is reduced during halothane anesthesia (Sonntag et al 1978)[71]. However, after 2 to 5 hours of constant halothane anesthesia all the cardiovascular changes (i.e., hypotension, depressed cardiac output, and bradycardia) tend to return toward normal; this response has been attributed to sympathetic activation with time (Eger et al 1970)[16].

Cardiac rhythm:

The heart rate is slow during anesthesia with halothane. This is, in part, reversible by atropine and is due to reduction of cardiac sympathetic activity with consequent vagal predominance. However, direct, atropine-insensitive slowing of SA nodal discharge can be observed in vitro; both a reduced rate of phase-4 depolarization and an increased threshold for the generation of an action potential appear to be involved. During halothane anesthesia, vagal activity is further enhanced by manipulation of the airway.

Halothane may also increase the automaticity of the myocardium; this effect is aggregated by adrenergic agonists and leads to propagated impulses from ectopic sites within the atria or ventricles.

Baroreceptor control:

Although early work demonstrated that halothane reduces afferent discharge by "resetting" the baroreceptors to respond around a lower "set point", depresses the vasomotor responds of the brainstem, and reduces the sympathetic outflow that results, the observed changes are small. In addition, halothane has little effect on the response of preganglionic symphathetic neurons to stimulation of baroreceptors (skovsted et al., 1969)[70]. Thus, it is concluded that the predominant actions of halothane are at the effector sites in the heart that controls cardiac rate and or contractility.

Organ Blood Flow:

Halothane influences the blood flow to every organ by both direct and indirect actions (Seyde and Longnecker, 1984) [68]. These include interference with the generation or action of factors derived from endothelium that regulate the tone of vascular smooth muscles (Stone and Johns,1989) [72]. In the skin and cerebral circulation, flow may increase as the vessels dilate. However, the cerebrovascular bed, as well as the renal and splanchnic circulations, loses some of its ability to autoregulate flow, and perfusion of these tissues decreases if blood pressure falls excessively. The coronary circulation remains responsive to myocardial needs for oxygen; vasodilation

occurs in poorly ventilated areas of the lung because of inhibition of pulmonary vasoconstriction that normally occurs in response to hypoxia.

In an individual patient, pH and carbon dioxide tension, posture, temperature, age, disease, and the administration of other drugs can influence blood flow to each of these organs.

Respiration:

If the patient anesthetized with halothane is allowed to breathe spontaneously, an increased partial pressure of carbon dioxide in the arterial blood is common and is indicative of ventilatory depression; there is also an increased difference between the partial pressure of oxygen in the alveolar gas and in the arterial blood, indicating less efficient exchange of gas. Halothane thus influences both ventilatory control and the efficiency of oxygen transfer. To compensate for these effects, ventilation is frequently assisted or controlled by manual or mechanical means, and the concentration of inspired oxygen is increased.

Ventilatory Control:

Characteristically respirations are rapid and shallow during halothane anesthesia. Minute volume is reduced, and arterial carbon dioxide tension is

increased from 40mm of Hg to approximately 50mm of Hg. Halothane causes a dose related reduction in the ventilatory response to carbon dioxide (Knill and Gelb,1978)[40].

Pulmonary Oxygen Transfer:

Efficient transfer of oxygen from the alveolar gas to hemoglobin in the alveolar capillary red blood cell depends on a proper balance between alveolar ventilation and perfusion. This balance is importantly conrolled by the effects of gravity and various structural mechanical factors, and fine adjustments are provided by changes in the tone of the smooth muscle of bronchial airways and pulmonary vessels. All these factors may be altered during halothane anesthesia.

Nervous System:

Electrical activity of the cerebral cortex recorded by a frontooccipital EEG shows progressive replacement of fast, low voltage activity by slow waves of greater amplitude as halothane anesthesia is deepened. Surgical stimulation may reverse this pattern, and such arousal reactions may be associated with recall of intraoperative events by patients, as in a dream (Bimar and Bellville,1977)[3].This sequence resembles arousal of the brain by

activation of brainstem reticular formation, but reticular neuronal activity is depressed by halothane (Shimoji et al., 1977) [69].

Since cerebral blood flow generally increases during halothane anesthesia, cerebrospinal fluid pressure increases (Lassen and Christensen, 1976)[41]. Halothane may thus aggravate conditions in which intracranial pressure is elevated. The cerebral metabolic consumption of oxygen is reduced and the delivery of oxygen and substrates to the brain appears to be adequate, but there are marked regional differences (Eintrei et al., 1985)[17]. After several hours of anesthesia with halothane the changes in the cerebral blood flow and metabolism return toward normal (Warner et al., 1985)[77].

Recovery of mental function after even brief anesthesia with halothane is not complete for several hours, but this phenomenon probably contributes little to the more prolonged impairment of psychological performance that has been reported after major surgery. Shivering during recovery is common and probably represents both a response to heat loss and an ill-defined expression of neurological recovery.

Muscle:

Relaxation of skeletal muscle is desirable or necessary for many surgical procedures. Anesthesia with halothane causes some relaxation by

central depression; in addition, the duration and magnitude of the muscular relaxation induced by non-depolarizing skeletal muscle relaxants such as tubocurarine or pancuronium are increased. The mechanism of this effect is not known but appears to be based on increased sensitivity of the end-plate to the action of the competitive neuromuscular blocking agents (Waund,1977)[78].

Rarely, induction of anesthesia with halothane or any of other halogenated inhalational anesthetics triggers an uncontrolled hypermetabolic reaction in the skeletal muscle of susceptible patients. Rapid rise in the body temperature and a massive increase in oxygen consumption and production of carbon dioxide characterize the resultant syndrome of malignant hyperthermia; death may result unless the anesthetic is discontinued and treatment with dantrolene is begun promptly. This syndrome may be caused by a defect in the uptake of Ca^{2+} by the sarcoplasmic reticulum in genetically susceptible muscle(Gronert,1980)[28].

Uterine smooth muscle is relaxed by halothane. This effect is of sufficient magnitude to allow manipulation of fetus during the pre natal period. Inhibition of natural or induced uterine contractions by halothane during parturition may prolong the process of delivery, as well as increase blood loss.

Kidney:

Halothane causes dose dependent reductions of renal blood flow and the rate of glomerular filteration; these parameters may be 40 to 50% of normal at 1 MAC (Mazze et al., 1963)[47]. Preoperative hydration and prevention of hypontension can attenuate these effects. Halothane does not interfere greatly with autoregulation of renal blood flow nor, in the normotensive state, with the distribution of flow between the renal cortex and medulla (Leighton and Bruce, 1975)[42]. Anesthesia is normally accompanied by the production of a small volume of concentrated urine. The changes in urine volume are probably secondary to circulatory responses and reduced glomerular filteration (Deutsch et al., 1966)[11]. The renal effects of halothane anesthesia are rapidly reversed, and there is no evidence of postoperatively results in hyponatremia, reduced plasma osmolality, and mental confusion.

Liver and Gastrointestinal Tract:

Splanchnic and, therefore, hepatic blood flow is reduced by halothane as a passive consequence of reduced perfusion pressure, but there is no evidence of overt ischemia (Epstein et al., 1966 [20]; Seyde and Longnecker,

1984)[68]. Hepatic cellular functions are, however, depressed, and halothane reduces the ability of the microsomal enzymes systems to metabolize drugs.

Bio-transformation:

Approximately 60 to 80% of absorbed halothane is eliminated unchanged in the exhaled gas in the first 24 hours after its administration, and smaller amounts continue to be exhaled for several days or even weeks. Of the fraction not exhaled, as much as 50% undergoes bio-transformation, and the rest is eliminated unchanged by other routes.

The mixed function oxidase or cytochrome P_{450} system in the endoplasmic reticulum of the hepatocyte is responsible for this bio-transformation. Chlorine and to a lesser extent bromine are removed from halothane; since the bond energy for C-F is nearly twice that for C-Br of C-Cl, little fluorine is removed [24].

Advantages:

A. Halothane is non inflammable and does not irritate the respiratory Passage. It has a fruity odour and is not unpleasant for induction.

B. Induction of anesthesia and recovery are reasonably quick. The incidence of postoperative vomiting is low.

C. Halothane inhibits laryngeal and pharyngeal reflexes in upper planes of surgical anesthesia to a considerable extent. It also relaxes the masseter muscles and inhibits salivation; hence tracheal intubation is much easier with this agent. It does not cause laryngospasm, bronchospasm, coughing, but produces bronchodilation by a direct relaxation of the bronchial smooth muscle. Hence it is preferred in-patients with history of bronchial asthma.

D. Halothane may be employed to induce control hypotension to provide a "bloodless" field during surgery but is safe in expert hands with Boyles anesthetic apparatus (Figure-IV).

Figure - IV

Disadvantages:

A. Muscular relaxation with halothane alone is inadequate to permit the surgical procedures; however, it potentiates the actions of d-tubocurarine, includeing its ganglionic blocking effect and this may lead to profound hypotension during anesthesia.

B. It depresses respiration if its concentration in the anesthetic vapour is allowed to exceed 2%.

C. It causes cardiovascular depression and hence, hypotension is a major drawback with halothane anesthesia. It exerts direct depresant action on the heart, decreases the cardiac output, reduces the sympathetic outflow and increases the parasympathetic tone. The total peripheral resistance changes very little even when hypotension occurs. With increased concentration it causes bradycardia. With rapid inhalation or a sudden increase in the concentration of halothane, the blood pressure may decrease suddenly and cardiac arrest may supervene.

Halothane sensitizes the ventricular muscle and conduction tissue to sadrenaline. It may increase automaticity of the myocardium and may cause arrhythmias; usually, these are benign.

D. Recovery of mental function after halothane takes several hours. Shivering during recovery is common.

E. Hepatic damage including extensive hepatocellular necrosis, due to allergy or idiosyncrasy. Individuals with hepatic disease and pregnant women are more likely to develop severe hepatotoxicity. Halothane should not be repeated at intervals of less than 3 months to avoid liver toxicity and should be avoided during pregnancy.

F. Halothane is a poor analgesic and must be supplemented with nitrous oxide or opiates to provide satisfactory conditions for operation.

G. Halothane causes rise in intracranial pressure due to cerebral vasodilation; hence, it is contraindicated in-patients with intracranial lesions[65].

Intravenous Anesthetics:

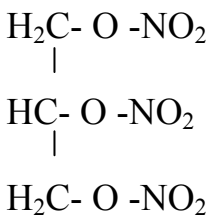
The intravenous anesthetics are:

1. Nitroglycerine
2. Esmolol.

1. Nitroglycerine:

The drug has been used for years to treat patient's coronary artery spasm or with myocardial infractions and to control periods of hypertension in-patients with known coronary artery disease.

Chemical name of nitroglycerine is 1.2.3 propanetriol trinitrate. The chemical structures of nitroglycerine are:



Properties:

Nitroglycerine is basically a vascular smooth, muscular relaxant. It dilates both arterial and venous beds, however, it has preferential effect on the post capillary vessels, including large veins to which it promotes peripheral pooling of blood and decreasing venous return to the heart leading to the reduction of left ventricular end diastolic volume (pre-load). Arterial relaxation produced by nitroglycerine leads to reduction in systemic vascular resistance and arterial pressure (after load). These effects of nitroglycerine produce reduction in the myocardial oxygen consumption or demand and thus a more favorable supply-demand ratio is achieved.

The recommended doses of intra venous nitroglycerine reduce systolic, diastolic and mean arterial pressure while maintaining effective coronary perfusion pressure, unless there is an excessive pain in blood pressure.

Intra venous nitroglycerine therapy reduces elevated central venous and pulmonary capillary wedge pressures, pulmonary vascular resistance and systemic vascular resistance. Heart rate is usually slightly increased, as a part of the reflex response to the fall in blood pressure. Cardiac index may be increased, decreased or unchanged. In the presence of elevated left ventricular filling pressure and systemic vascular resistance along with a depressed cardiac index there is a likelihood of an improvement in cardiac index. On the other hand, when filling pressures and cardiac index are normal, cardiac index may be slightly reduced by intra venous nitroglycerine therapy.

Pharmacokinetics:

Nitroglycerine is widely distributed in the body and is rapidly metabolized to dinitrates and mononitrates. It has a short half-life, estimated of 1 to 4 minutes. This makes it possible to monitor and control the plasma levels of nitroglycerine during intra venous infusion.

Nitroglycerine is readily absorbed into the plastic materials parenteral solution containers and tubings of intra venous infusion sets. Therefore, the dilation and storage of nitroglycerine for intra venous infusion should be made only in glass parenteral solution bottles.

Forty percent to eighty percent of the total amount of nitroglycerine in the final diluted solution for infusion is absorbed by the polyvinyl chloride (PVC) tubing of the intra venous administration sets. The rate of absorption depends upon the flow rate, nitroglycerine concentration and the length of the tubing, etc.,.

Indications:

1. Angina pectoris, including unstable, angina pectoris and post-infraction angina pectoris in-patients who have responded to recommended doses of organic nitrates and or beta-blockers.
2. Acute myocardial infraction, congestion heart failure and improvement of coronary circulation opening of collaterals and reduction of infract size.
3. Control of blood pressure in perioperative hypertension i.e., hypertension associated with surgical procedures, especially cardiovascular procedures, such as hypertension seen during intratracheal intubation, anesthesia; skin incision, stemotomy, cardiac bypass and in the immediate post-surgical period.
4. Production of controlled hypotension during procedures.

Contraindications:

1. Known hypersensitivity to nitroglycerine or other organic nitrates.
2. Hypotension or uncovered hypovolaemia as the use of nitroglycerine injection in such conditions could produce severe hypotension or shock.
3. Increased intracranial pressure (e.g: head trauma or cerebral hemorrhage.
4. Inadequate cerebral circulation, constrictive pericarditis and cardiac tamponade.

Precautions:

1. Nitroglycerine injection should be used with caution in-patients with severe hepatic or renal disease.
2. Excessive fall in blood pressure, especially for prolonged periods of time, must be avoided because of possible deleterious effects on the brain, heart, liver and kidney from poor perfusion and the attendant risks of ischaemia, thrombosis and altered functions of these organs. Patients with normal or low pulmonary capillary wedge pressure are especially sensitive to the hypotensive effects of nitroglycerine injection. It is advisable to monitor the pulmonary

capillary wedge pressure to titrate the dosage of the drug and as it generally precedes the occurrence of arterial hypotension.

3. Use during pregnancy: The safety of the use of nitroglycerine injection during pregnancy has not been established, therefore it should be given to pregnant women only if clearly needed.

Adverse effects:

The commonly observed adverse effects of nitroglycerine injection are extensions of its pharmacological actions, which include headache, flushing, tachycardia, palpitations, retrosternal discomfort and dizziness. Nausea, vomiting and abdominal pain have also been reported. Paradoxial bradycardia and increased angina pectoris may accompany nitroglycerine-induced hypotension.

Dosage and administration:

Nitroglycerine injection is a concentrated, potent drug, which must be diluted in 5% dextrose or normal saline prior to its infusion. It should not be admixed with other drugs.

The administration of nitroglycerine infusion is initially begun with 5 microgram per minute with subsequent titration to adjust that dose in accordance with the clinical situations with increments of 5 microgram per minute, every 3 to 5 minutes until some response is noted. If no response is seen at 20 microgram per minute, increments of 10 and later 20 microgram per minute can be used. Once a partial blood pressure response is observed, the dose increase should be reduced and the interval between increments should be lengthened.

Esmolol:

Esmolol is selective β_1 antagonist with a very short duration of action. It has little if any intrinsic sympathomimetic activity and it lacks membrane stabilizing actions. Esmolol is administered intravenously and is used when β blockade of short duration is desired or in critically ill patients in whom adverse effects of bradycardia, heart failure, or hypotension may necessitate rapid withdrawal of drug.

Absorption,fate and excretion:

Esmolol has a half-life of about 8 minute and an apparent volume of distribution of approximately 2 litres/Kg. The drug contains an ester linkage,

and it is hydrolyzed by esterases in erythrocytes. The half-life of carboxylic acid metabolic of esmolol is far longer (4 hours), and it accumulates during prolonged infusion of esmolol. However, this metabolite has very low potency as a β-adrenergic antagonist (Reynolds et al., 1986)[63]; it is excreted in the urine.

The onset and offset of β-adrenergic blockade with esmolol are rapid; peak hemodynamic effects occur within 6 to 10 min of administration of a loading dose, and there is substantial attenuation of β blockade within 20 minutes of stopping an infusion. Esmolol may have striking hypotensive the effects in normal subjects, although the mechanism is uncertain (Reilly et al., 1985)[61].

Preparations, Routes of Administration and Dosage:

Esmolol hydrochloride is available in a concentrated solution (250 mg/ml) to be diluted prior to use, or in a solution (10 mg/ml) for intravenous administration. To initiate treatment, a loading dose of 500 μg/kg is infused over 1 minute; this is followed by infusion of 50 μg /kg / minute for 4 minutes. If an adequate therapeutic effects is not observed within 5 minutes, the same loading dose is repeated and maintenance infusion is increased to 100μg /kg/minute. The titration can be repeated with progressively greater 4-

Minute infusions. As the desired endpoint is approached, the loading dose is omitted and the increment in the maintenance infusion is made more gradually. Responses to esmolol usually occur with maintenance rates above 50 to 200µg / kg /minute; the safety of maintenance rates above 300µg/ kg /minute is not known. Infusions have been tolerated for up to 48 hours.

Effect of hypotension on organ function

Deliberate hypotension decreases arterial pressure; it provides the surgeon with a dry field and decreases the need for blood transfusion the mechanism responsible for the decrease in arterial blood pressure are diverse and exert different influences on the various organs of the body. Obviously deliberate hypotension is designed to decrease arterial blood pressure while preserving organ blood flow and thereby preserving organ function. It must be emphasized that decreasing blood pressure by hemorrhage diminishes organ blood flow. A deliberate hypotension technique requires that intravascular fluid volume be constantly judged throughout the surgical procedure so as to ensure optimal organ function.

The effects of hypotension on various organ beds are complex, depending on the drugs used and the magnitude and duration hypotension.

Skin and Muscle:

Hoffman et al [33]., found that in rats blood flow to skin decreased with sodium nitroprusside, nitroglycerine, and 2% enflurane while skeletal blood flow increased.Wild smith et al[79]., studied 20 patients undergoing middle ear surgery who received either sodium nitroprusside or trimethaphan to induce

hypotension. These workers found no change in lactate, pyruvate,or standard bicarbonate with either drug. Extensive clinical experience with a variety of drug without evidence of myoglobinuria, skin necrosis, or muscle weakness would support the claim that deliberate hypotension is not injurious to these tissues. However, Hauss et al[31]., found that in the dog, skeletal muscle PO_2 was markedly lowered by sodium nitroprusside and not by nitroglycerine. These investigators suggested that the autoregulation of the microcirculation is severely impaired with nitroprusside but not with nitroglycerine.

Endrich et al [19]., compared the effects of nitroprusside and nitroglycerine on striated muscles of hamsters. They not only examined tissue oxygenation but also measured microvascular pressures, vascular diameters, and density, and blood cell velocity and directly visualized the microvasculature. Significant differences between the two agents were seen. Both agents affected resistance vessels similarly, but post-capillaries dilated in response to nitroglycerine. Tissue oxygenation was decreased by sodium nitroprusside but not by nitroglycerine. They concluded that nitroglycerine may have a distinct advantage over sodium nitroprusside. Unfortunately, the desired degree of hypotension with nitroglycerine cannot always be achieved.

Central Nervous System:

When deliberate hypotension is used, the main concerned is whether cerebral perfusion is adequate. Adequacy of cerebral blood flow suring controlled hypotension has been assessed by the following methods.

a. Level of consciousness

b. Jugular bulb venous oxygen tension.

c. Electroencephalography (E.E.G)

d. Higher cerebral function after surgery

e. Cases with cerebral damage after operation

f. Bichemical indices

g. Radioactive xenon clearance.

The current rationale for the safe lower limit of mean arterial blood pressure of 50 to 55 mm of Hg in normothermic patients is based on the concept that the lower limit of autoregulation for cerebral blood flow is at this range. Michenfelder and Theye [49] saw clear neurologic deflicits in two of 5 dogs anesthetized with nitrous oxide and subjected to 1 hour of hypotension at a mean arterial pressure of 40-mm Hg by trimethaphan. Dong et al., [12] demonstrated that in dogs , there was a poor correlation between EEG and brain function. They subjected dogs to profound hypotension with trimethaphan for one hour. One half the animals died, but this was due to

myocardial and liver damage, whereas the surviving animals showed no neurologic changes. Pinaud et al [57]., recently studied 9 adult patients undergoing for repair of cerebral aneurysms. Mean arterial pressure was lower to 40 mm of Hg with sodium nitroprusside. Intraoperative cerebral blood flow and cerebral metabolic rate for oxygen were measured before, during, and after hypotension. They concluded that this reduction of mean arterial pressure was apparently safe for the reason of the brain studied. In poorly perfused regions, local brain and cerebrospinal fluid lactoacidosis occurring especially in the retracted areas might cause at these low pressures, but the study technique did not allowq for such a determination.

Ishikawa and McDowall [35] examined the affects of nitroprusside and trimethaphan in cats subjected to mean arterial pressures of 30,35and 40 mm Hg. Their data demonstrated that cerebral blood flow was significantly greater with sodium nitroprusside than with trimenthaphan.

Cerebral blood flow appears to be maintained at adequate rates to supply the needs for the brain at a mean arterial blood pressure of 50 to 65 mm Hg. Grubb and Raichle [29] found that cerebral blood flow is actually increased with sodium nitroprusside and reasoned that it might be due to stimulation of the sympathetic nervous system with release of circulatory catecholamines. Adeeper levels of hypotension with sodium nitroprusside may be preferable.

Other hand cerebral perfusion, the effect of drugs on intracranial pressure is also of concern, especially in-patients with elevated intracranial pressure. In examination the influence of trimethaphan induced hypotension in cats, Stulken and Sokoll [73] found significant increases in intracranial pressure as compared with hypotension induced by sodium nitroprusside. Subsequently, several investigators found that nitroglycerine , sodium nitroprusside and verapamil allincreased intracranial pressure. Thus, these drugs should not be used until the cranium is opened in a patient with suspected increased intracranial pressure. If the measurement of intracranial pressure is available before the surgical procedure, titration of drug before the opening of the skull seems justified.

Eye:

The intraocular pressure of the eye is due to blood and aqueous humor. With a reduction in arterial blood pressure, intraocular pressure decreases. The eye has two separate systems of blood vessels, the retinal and uveal. The uveal vessels are peculiar in that they possess no precapillary sphincters, hence a steady blood flow. Since the uveal systems carries the major blood pressure are transmitted to the eye with a resultant decrease in intraocular pressure. Because of the effect of hypotension on blood flow to the eye, some

of the complications of deliberate hypotension are blurring of vision and rarely blindness.

Heart:

The severe depression of the myocardium caused by deep halothane anesthesia was a major factor in the development of intravenous hypotensive agents. A wealth of information comparing the effects of nitroprusside, nitroglycerine, and trimethaphan used to treat hypertension intraoperatively. Only a few studies have examined the effects of these agents on the myocardium when mean arterial blood pressure has been decreased to 50 to 65 mm Hg. Vance et al [76]., suggested that during hypotension induced by nitroprusside, coronary blood flow is well maintained. Jupa et al [36]., compared sodium nitroprusside and trimethaphan and demonstrated that auto-regulation of coronary blood flow was better maintained with sodium nitroprusside. For example, some patients such as those undergoing clipping of the cerebral aneurysm , may require hypotension to help in the surgical procedure. These same patients may also have underlying coronary artery disease. Therefore, knowledge of the use of these drugs during deliberate hypotension is important.

Hickey et al [32]., examined the effects of deliberate hypotension in a dog model with a single coronary artery stenosis. Hypotension was induced with

trimethaphan, halothane, or sodium nitroprusside to a MAP of 50 mm Hg. In the normal portions of the myocardium, trimethaphan and halothane significantly reduced subendocardial and subepicardial blood flow. A similar reduction in myocardial oxygen consumption occurred for each agent. This suggests that coronary blood flow is dependent on myocardial metabolic demands. By contrast, sodium nitroprusside did not affect regional coronary blood flow or myocardial oxygen consumption. With a critical stenosis, hypotension with all drugs resulted in reduced regional myocardial blood flow and a lowered perfusion pressure. However, myocardial ischemia did not occur. Lactate extraction continued and no changes in St segments and no redistribution of transmural blood flow were found. With several stenosis, deliberate hypotension reduced perfusion pressure, depressed ST segments, and produced evidence of ischemia. The date suggested that trimethaphan was the least injurious, but the number animals studied was small. It should be emphasized that this was a dog model and that the stenosis involved only one vessel; this is not the common clinical finding in humans. Whether these findings apply to the patient with stenosis of several coronary arteries has not been investigated.

The use of isoflurane with or without other hypotensive agents has gained popularity. Decrease in mean arterial pressure to 50 to 60 mm Hg or primarily due to decrease in systemic resistance with minimal effects on cardiac output. Priebe [59] has shown, however, that there is some degree of

myocardial depression at 1.8% inspired isoflurane in dogs. This study demonstrated a dose dependent decrease in systemic vascular resistance and an even greater decrease in coronary vascular resistance.

Manninen et al [45]., studied 33 neurosurgical patients in whom hypotension to 50 mm Hg was achieved by using isoflurane anesthesia. Although they found that over 50% of the patients developed nonspecific ECG changes, there was no evidence of myocardial infraction when cardiac enzymes were examined.

Reiz et al [62]., first suggested that the coronary vasodilation seen with isoflurane may not necessarily be beneficial. The concept that such vasodilation could lead to regional myocardial ischemia was referred to as "coronary steal."

Adenosine is also a potent coronary dilator. Bloor et al [5] ., compared adenosine to sodium nitroprusside in 12 normal dogs. They found that a 50% reduction in mean arterial pressure by either agent resulted in morbid differences. Adenosine resulted in a marked increase in coronary blood flow, no increase in plasma catecholamines, and no change in cardiac lactic acid uptake. In contrast, sodium nitroprusside resulted in an increase in heart rate and plasma catecholamines with evidence of lactic acid production. Coronary sinus blood flow did not increase. Owell et al [55]., administered adenosine to

five neurosurgical patients and found significant increase in coronary blood flow together with decreases in myocardial oxygen consumption. They also found that the hypotension was easily controlled, probably because of inhibition of both the sympathoadrenal and the renin-angiotensin systems. Presently, with the limited data available, adenosine can be safely used in-patients without coronary artery disease. The potential benefits of adenosine in-patients with coronary disease must be tested clinically.

Lungs:

Eckenhoff et al [13]., noted a higher than normal carbon dioxide tension in the blood during deliberate hypotension using either pentolinium or trimethaphan. In measuring physiologic dead space in 25 patients, they concluded that hypotension combined with increased mean airway pressure, head-up tilt, and surgery all may lead to increased dead space. Khambatta et al., [38] measured dead space in-patients undergoing hypotensive anesthesia with sodium nitroprusside. They concluded that if cardiac output was maintained by replacement of intravascular fluids when hypotension was induced, there was no increase in physiologic dead space. This is supported by the findings of Suwa et al [74]., who demonstrated that a decrease in cardiac output would result in an increase in dead space; this may explain the findings reported by Echenhoff et al [13] .

Oxygenation during deliberate hypotension may also be altered. Wildsmith et al [80]., demonstrated a marked decrease in arterial oxygenation of sodium nitroprusside. In-patients with chronic obstructive pulmonary disease (COPD) in whom the shunt fraction was already increased, no change in shunt fraction could be demonstrated. Nitroglycerine infused both in normal patients and in those with COPD responded in a manner similar to that found with sodium nitroprusside. Bernard et al [2]., studied 16 patients undergoing total hip arthoplasty. Deliberate hypotension was induced with either sodium nitroprusside or isoflurane. Pulmonary shunt fraction increased with sodium nitroprusside but not with isoflurane.

Other effects of hypotensive agents on pulmonary function are minimal. The trimethaphan also causes a slight increase in respiratory rate as well as slight alveolar hyperventilation. Lung mechanics was unchanged. Because of changes in oxygenation and possibly carbon dioxide elimination, controlled ventilation is preferred for patients undergoing deliberate hypotension.

Kidneys:

Although renal blood flow is decreased, untoward effects are not evident. Behnia et al [1]., examined the effect of sodium nitroprusside on humans. Arterial blood pressure was reduced to 50 mm Hg and creatinine

clearance determined. Mannitol, 1 to 1.5gm/kg, was infused to maintain urine flow at 1 to 2ml during in the entire procedure. Creatinine sclearance decreased significantly only during the period of hypotension but rapidly return to control when arterial blood pressure returned to normal. In trimethaphan, urine flow was maintained greater than 2ml/minute by the infusion of 5% dextrose in lactated Ringer's solution at 10ml/kg/hour. These studies also showed a transient decrease in renal function during the period of hypotension.

Dong et al [12]., found no increase in blood urea nitrogen (BUN) is creatinine in dogs subjected to severe hypotension. Thompson et al [75]., could find no significant changes in serum creatinine, BUN, serum or urinary electrolytes in 30 patients having deliberate hypotension. Since renal dysfunction is not a frequent complication of deliberate hypotension, short periods of decreased renal flow apparently are not detrimental.

Splanchnic circulation:

One of the most difficult areas to monitor clinically is the liver circulation, as portal venous circulation has no significant autoregulation and hepatic autoregulation is minimal. Thus liver damage could be severe during deliberate hypotension. With MABP of 12 to 25mm Hg during trimethaphan induced hypotension, severe disturbances in liver enzymes result, and hepatocyte degeneration and areas appear with pericentral lobular mecrosis.

When lesser degrees of hypotension are employed, a mild elevation in bromsulfalien retention was noted. Other tests of liver function showed no significant changes.

The effect of isoflurane on the splanchnic circulation is complex, Conzen et al., [8] studied liver blood flow and oxygen tensions in the liver of dogs anesthetized with isoflurane. With increasing amounts of isoflurane there was a dose dependent decrease in liver blood flow because of a decrease in portal flow. Splanchnic oxygen consumption remained unchanged. This resulted in a significant decrease in hepatic surface oxygen tension.

Complications:

Surgical Comomplications: Reactive hemorrhage

Cut vessels may not bleed during intraoperative hypotension. When the arterial pressure increase after operation, bleeding may begin. Reactionary hemorrhage and hematoma function have not been a problem, as bleeding can usually be prevented simply by the application of pressure dressings, and by the avoidance of sudden return to higher pressures following surgery.

Stasis is not associated with low pressures produced by vasodilating techniques. There is no evidence that the incidence of venous thrombosis is increased.

Collection of Data

DATA COLLECTION

Preoperatively the gauze was cut into pieces of 4X6 inches and the weight of each gauze was determined(Fig.V). The length and weight of throat pack gauze (Roller gauze) was measured. The number of gauzes used for the surgery and the total weight of the gauze was determined, by using the common balance. All the gauzes were sterilized after measuring.

Figure - V

A total number of 40 gauzes were prepared for every surgery and the weight of 40 gauzes were noted. For every 15 minutes the surgeon Fromme's ordinal scale assessed the surgical field.

1)By the surgeon's comment based on Fromme's Ordinal Scale of Assessment of Surgical Field is as follows.

5-Massive uncontrollable bleeding

4-Bleeding, heavy but controllable, that significantly interfered with the dissection.

3-Moderate bleeding that moderately compromised surgical dissection.

2-Moderate bleeding, a nuisance but without interference with accurate dissection.

1-Bleeding, so mild that it was not even a surgical nuisance.

0-No bleeding, virtually bloodless field.

All these readings were recorded for every 15 minutes. After the surgery was completed, the volume of fluid in the suction apparatus was measured (Fig.VI). The number and the weight of the used gauze were measured postoperatively using common balance and also the weight of unused gauze was measured.

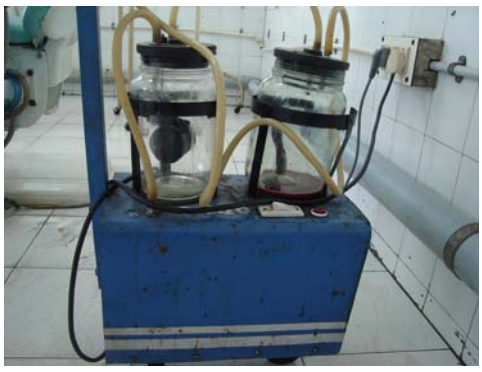


Figure - VI

The blood loss was assessed by

a) Calculating the difference in weight of the gauze, both pre and Post-operatively(Fig.VII).

Figure – VII

b) Suction bottle volume minus irrigation fluid volume.

At the end of the surgery, all the patients were kept in the recovery room until fully awake and then shifted to the ward. Postoperative I V fluids were given depending upon the patient's weight and requirement. Urea, creatinine, serum bilirubin, Hb% and packed cell volume were done on the Ist postoperative day to know about the organ function.

Proforma

PROFORMA

DATE:

NAME:

AGE:

SEX:

O.P.NO:

PROCEDURE PLANNED:

PRE-OPERATIVE ASSESMENT:

Exclusion criteria (ASA-1,patients only are accepted for the study)

1.Cardiac disease

2.Bronchial asthma (Allergic-cannot use β-blockers)

3. Hypertension

4. Pre-op hypotension

5. Liver dysfunction

6. Renal dysfunction

PRE-MEDICATION:

PRE-OPERATIVE B.P:

PRE-OPERATIVE HB%: POST-OPERATIVE HB%:

INTRA-OPERATIVE MONITORING:

STARTING TIME FINISHING TIME

SURGERY

ANESTHESIA

ANESTHESIA DRUGS USED:

1.

2.

3.

4.

5.

6.

7.

BLOOD LOSS ASSESSMENT

Amount of saline used (Total for irrigation)

Volume collected in the suction apparatus:

No.of small gauze used:

No. of large gauze used (Throat pack):

Fluids given intra-venously at O.T:

Estimation of blood loss by surgeon:

Estimation of blood loss by anesthetist:

Total blood loss:

ASSESMENT CHART

TIME	BLOOD PRESSURE			HEART RATE	SPO_2	PROCE - DURE	DRUG ADMINISTERED	FLUID ADMINISTERED	S US IRR
	SYSTOLIC	DIASTOLIC	MEAN						

RESULTS

NORMAL GROUP

S.N	AG	SE	LENGTH OF SURGERY	LENGTH OF ANESTHESIA	EBL BY SURGEON	EBL BY ANESTHETIST
1	24	Fem	2Hrs.	2Hrs 15 mts.	900ml	1000ml
2	21	Fem	2Hrs. 30 mts.	2Hrs 45 mts.	700ml	800ml
3	34	Fem	2Hrs. 20 mts.	2Hrs 40 mts.	700ml	800ml

Table – 1 showing age, sex, length of surgery, length of anesthesia, estimated blood loss by surgeon and anesthetist in control group

HYPOTENSION GROUP

S.N	AG	SE	LENGTH OF SURGERY	LENGTH OF ANESTHESIA	EBL BY SURGEON	EBL BY ANESTHETIST
1	22	Mal	3 Hrs. 30 mts.	3Hrs. 40 mts.	600 ml	575ml
2	22	Fem	2 Hrs. 30 mts.	2Hrs. 40 mts.	200 ml	150ml
3	25	Fem	2 Hrs. 45 mts.	3Hrs. .	500 ml	545ml

Table – II showing age, sex, length of surgery, length of anesthesia, estimated blood loss by surgeon and anesthetist in
hypertension group

\

NORMAL GROUP

S.No.	15 min	30 min	45 min	1 hr	1hr 15min	1hr 30 min	1 hr 45 min	2 hrs	2hr15m	2hr30m	2hr45m	3 hrs
1	1	1	2	1	1	3	1	0				
2	1	1	2	3	2	1	1	1	1	0		
3	1	1	3	2	1	1	1	1	0			

Table – III showing the scoring of the surgical field according to Fromme's ordinal scale in the control group.

HYPOTENSION GROUP

S.No.	15 min	30 min	45 min	1 hr	1hr 15min	1hr 30 min	1 hr 45 min	2 hrs	2hr15m	2hr30m	2hr45m	3 hrs
1	1	1	0	0	1	0	0	1	0	0	0	0
2	1	1	0	0	0	1	0	0	0	1		
3	1	1	0	0	0	0	1	0	0	0	1	

Table – III showing the scoring of the surgical field according to Fromme's ordinal scale in the Hypotensive group.

TABLE – V

VARIABLES	AGE	
CONTROL	MEAN	S.D
	26.3	5.26
HYPOTENSION	23.30	1.552

Table – V shows that the age distribution among the patients it reveals that the mean age is 26.30 with a standard deviation of 5.26 in the control group. In the hypotension group the mean age is 23.3 with a standard deviation of 1.55 This figure explains there is no difference between the ages for the control and hypotension group.

TABLE VI:

SEX	CONTROL		HYPOTENSION	
	No	**%**	**No**	**%**
Male	0	0	1	33
Female	3	100	2	66

Table VI shows that sex distribution among the patients. The Chi-Squared Value is 0.22. There is no association between the control and hypotension group With their sex.

TABLE VII:

VARIABLES	CONTROL		**HYPOTENSION**		t-VAUE
	MEAN	**S.D**	**MEAN**	**S.D**	**(P-VALUE)**
EBL by Surgeon	**733**	57.37	**433.3**	207.99	t.2.406 p<0.07
EBL by Anesthetist	**866**	122.61	**423**	427.87	t.1.74 p<0.16

A comparison of estimated blood loss (EBL) by the surgeon and anesthetist between control and hypotension group. The statistical analysis of the data by student t-test explained that there is a significant difference of EBL by surgeon and anesthetist between hypotension and control group at P<0.16 level. The mean values of EBL by surgeon and EBL by anesthetist in hypotension group were then compared with control group.

FIGUREVIII : COMPARISON OF ESTIMATED BLOOD LOSS BY SURGEON AND ANESTHETIST BETWEEN HYPOTENSION AND CONTROL GROUP

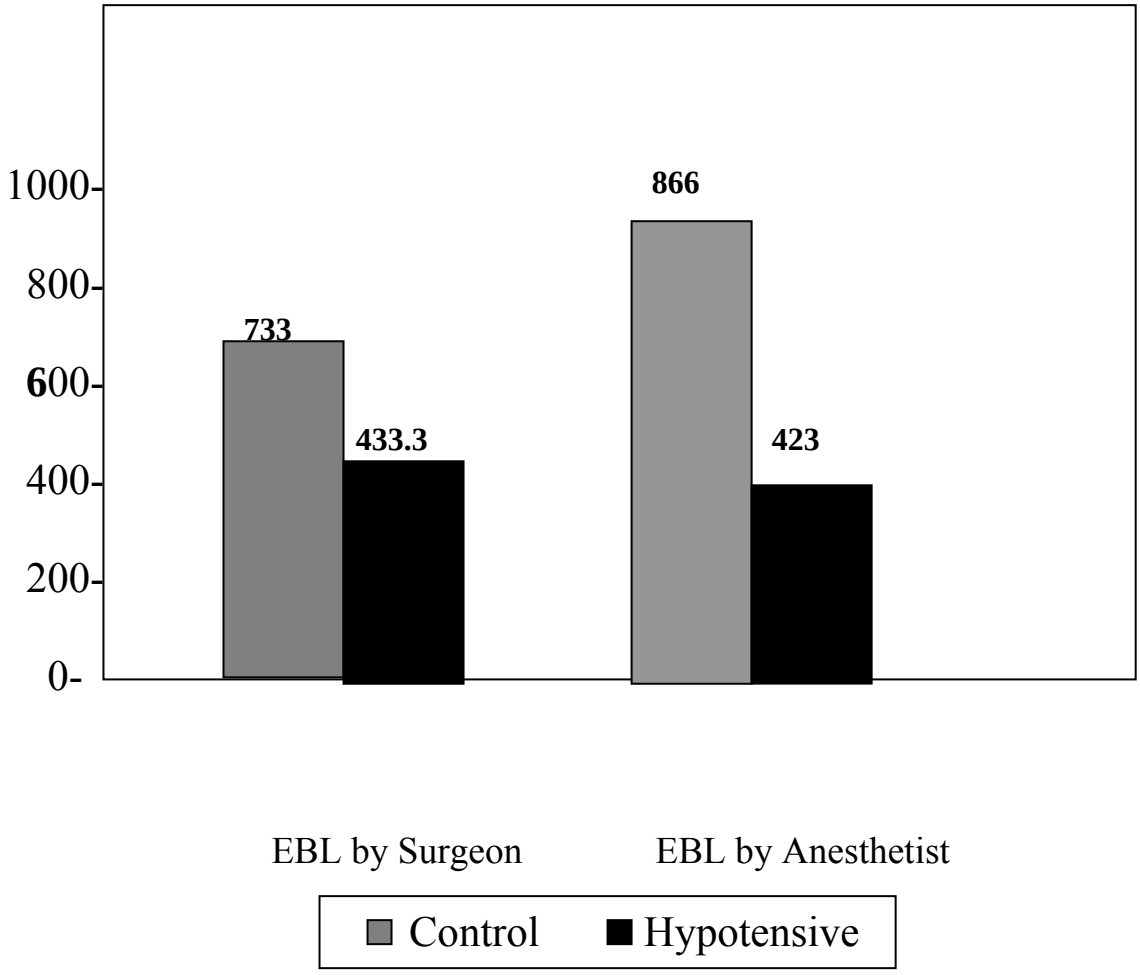


Fig. VIII is explain that the estimated blood loss by surgeon and Anesthetist shows the better values in hypotension anesthesia

TABLE VIII:

GROUP	MEAN	S.D
Control	1.25	0.04
Hypotension	0.37	0.02
t- Value	32.869 ($p<0.001$)	

Table VIII shows that the assessment of hypotension and control Group, the student t-test value is 32.869. It reveals that there is statistically Significant difference between hypotension and control group at $p<0.001$ Level. The assessment of the surgical field by surgeon reveals that the hypotension group is better than the control group.

PHOTOGRAPHS

A. NORMAL

1. NAME: MISS. VIDHYA, 21/FEMALE, I.P. No.84451
   Le Fort I Osteotomy

   PREOPERATIVE POSTOPERATIVE


2. NAME: MISS. NIRANJANI, 24/FEMALE, I.P. No.11561
   Anterior Maxillary Osteotomy

   PREOPERATIVE POSTOPERATIVE


3. NAME: Mrs. RANI, 34/FEMALE, I.P. No. 74822
   Anterior Maxillary Osteotomy

   PREOPERATIVE POSTOPERATIVE


PHOTOGRAPHS

B. HYPOTENSION

1. NAME: Mr. RAJA, 22/MALE, I.P. No. 58497
   Le Fort I Osteotomy

PREOPERATIVE

POSTOPERATIVE

2. NAME: MISS. RUKMANI, 20/FEMALE, I.P.No.90029
   Anterior Maxillary Osteotomy

PREOPERATIVE

POSTOPERATIVE

3. NAME: MISS. KAVITHA, 25/FEMALE, I.P. No.63049
   Anterior Maxillary Osteotomy

PREOPERATIVE

POSTOPERATIVE

discussion

DISCUSSION

This investigation demonstrated that induced hypotension anesthesia patients in our group did result in reduced blood loss (as measured by the three methods).

1. Estimated blood loss by surgeon
2. Estimated blood loss by anesthetist.'
3. Fromme's ordinal scale for assessment of surgical field and improved surgeon's assessment of the surgical field.

Kelly and Terry[37] assessed intravascular volume changes in orofacial corrective changes using chromium-51, double tagged red blood cells and radioiodinated serum albumin for red blood cell volume and plasma volume, respectively and concluded that there was no correlation between the surgeons estimated of blood loss and either the isotope measured changes. Of the three techniques of estimated of blood loss used in one study, as one might suspect because a similar study was done by Precious D.L et al[58] which showed that the surgeons estimated tended to lie towards the lower estimated when reviewing with the data of Kelly and Terry[37].

One might question, the use and accuracy of the non-invasive blood pressure monitor (NIBP) for a study of this nature. Certainly, the "gold standard" of blood pressure measurement is an intra-arterial catheter transducer system. These are however, not without risk. The accuracy of automatic non-invasive blood pressure monitors has been questioned. The unit used in our study was Hewlett Packard M3046A monitor cycling every 10 minutes. In our study, blood pressure was lowered to a level significantly different from that of the control group.

The Fromme's ordinal scale was used as a tool of "surgical field assessment" in a reasonable attempt to objectively standardize this difficult to assess subjective parameter.

We could foresee some potential bias, dependent on the difficulty of surgery, included in each group despite the effort to block and stratify the surgical cases.

Contraindications to the use of induced hypotension anesthesia often relate to preexisting organ perfusion and oxygen delivery, but these problems are not typically or routinely found in our study population.

Thompson et al[75] demonstrated that there was no difference in organ function indices between a control group and an induced hypotensive group during total hip arthroplasty, our study also did not lead to any organ dysfunction in either of the groups postoperatively.

McNulty et al[48] showed no adverse clinical sequelae after induced hypotensive anesthesia using labetolol in 10 orthoganthic surgery patients. Similarly, no significantly complications were sited in our study using various methods of induced hypotensive anesthesia i.e., nitroglycerine, esmolol and halothane.

In our study 6 orthognathic surgery patients were studied to compare blood loss, surgical field quality and procedure duration with and without induced hypotension anesthesia. Precious et al [58], in a similar study showed that induced hypotension anesthesia reduced blood loss, and improved the surgical field, but did not significantly shorten the procedure. Our study revealed that induced hypotensive anesthesia, resulted in reduced blood loss and improvement in surgical field which was similar to Precious et al [58] but the time of surgical procedure showed a statistical difference between the two groups and the mean time of surgery in the induced hypotension anesthesia group was less when compared to the normotensive control group. This could be due to less surgical field and better visualization to work faster.

SUMMARY

SUMMARY

A study was concluded on 6 patients undergoing osteotomy procedures for facial correction. The study population consisted of two groups. One group of 3 patients (normotensive anesthesia group) and the other group of 3 patients (hypotension anesthesia group), a comparative study on blood loss intra-operatively in these two groups were studied by assessing and comparing the blood loss estimated by the surgeon, anesthetist and surgical field.

The drugs used for anesthesia for the normotensive groups were nitrous oxide and halothane and for the hypotensive group the addition of nitroglycerine and esmolol. Blood loss was evaluated by the surgeon, anesthetist and according to Fromme's scale of surgical grading at intervals of 15 minutes from the onset of procedure to the end of the procedure.

Our study showed that there is a significant difference between the blood loss in both the groups, and induced hypotension anesthesia group

showed reduced blood loss as assessed by the surgeon, anesthetist and according to Fromme's scale. There was neither any organ dysfunction due to tissue hypoxia in any one of the subjects nor any complications.

conclusion

CONCLUSION

Technique for minimizing blood loss are important for reducing the need for transfusion and the potential risk of a transfusion reaction or the transmission of blood borne pathogens. This is especially relevant when considering the elective nature of orthognathic surgery. Induced hypotensive anesthesia is a useful and safe technique in the orthognathic population when properly applied and executed.

Further well-designed, prospective investigations are needed to define, which agents are most efficient and safe; the recommended parameters of induced hypotensive anesthesia with respect to blood pressure reduction; and effect on blood loss using most accurately available qualifying technique.

Induced hypotension anesthesia not only reduced the blood loss, but also provides a blood loss field helping the surgeon to work more effectively and minimize the time of surgery.

Hence, induced hypotensive anesthesia should routinely be applied especially in elective surgeries like orthognathic surgeries, which is of high beneficial value.

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